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(FILE 'HOME' ENTERED AT 13:06:13 ON 24 JUN 2003)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 13:06:25 ON 24 JUN 2003

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594186	FILE GENBANK
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760	FILE PHIN

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             7   FILE RDISCLOSURE
        214895   FILE SCISEARCH
           102   FILE SYNTHLINE
         95818   FILE TOXCENTER
         33349   FILE USPATFULL
           829   FILE USPAT2
            67   FILE VETB
          1119   FILE VETU
          7215   FILE WPIDS
          7215   FILE WPINDEX
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     FILE 'BIOSIS, SCISEARCH, CAPLUS, MEDLINE, EMBASE' ENTERED AT 13:07:21 ON
     24 JUN 2003
L2          123 S L1 AND (SERUM GLUCOCORTICOID)
L3           54 S L2 AND HUMAN
L4           20 S L3 AND (ISOLAT? OR PURIF? OR CHRACTER? OR CLON?)
L5           11 DUP REM L4 (9 DUPLICATES REMOVED)
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=> d l5 ibib ab 1-11

L5 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:97550 CAPLUS
DOCUMENT NUMBER: 138:164674
TITLE: Molecular markers for hepatocellular carcinoma and their use in diagnosis and therapy
INVENTOR(S): Debuschewitz, Sabine; Jobst, Juergen; Kaiser, Stephan
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003010336	A2	20030206	WO 2002-EP8305	20020725
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
DE 10136273	A1	20030213	DE 2001-10136273	20010725

PRIORITY APPLN. INFO.: DE 2001-10136273 A 20010725
AB The invention relates to mol. markers occurring for hepatocellular carcinoma. The invention more particularly comprises gene sequences or peptides coded thereby which can be regulated upwards or downwards for hepatic cell carcinoma (HCC) in relation to healthy, normal liver cells in the expression thereof. The invention also relates to the use of said sequences in the diagnosis and/or therapy of HCC and for screening purposes in order to identify novel active ingredients for HCC. The invention also relates to an HCC specific cluster as a unique diagnostic agent for HCC.

L5 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:218843 CAPLUS
TITLE: Dehydroepiandrosterone affects the expression of multiple genes in rat liver including 11.beta.-hydroxysteroid dehydrogenase type 1: A cDNA array analysis
AUTHOR(S): Gu, Shi; Ripp, Sharon L.; Prough, Russell A.; Geoghegan, Thomas E.
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, The University of Louisville School of Medicine, Louisville, KY, USA
SOURCE: Molecular Pharmacology (2003), 63(3), 722-731
CODEN: MOPMA3; ISSN: 0026-895X
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Dehydroepiandrosterone (DHEA) is a C-19 adrenal steroid precursor to the gonadal steroids. In **humans**, circulating levels of DHEA, as its sulfated conjugate, are high at puberty and throughout early adulthood but decline with age. Dietary supplementation to maintain high levels of DHEA

purportedly has beneficial effects on cognitive memory, the immune system, and fat and carbohydrate metab. In rodents, DHEA is a peroxisome proliferator that induces genes for the classical peroxisomal and microsomal enzymes assocd. with this response. These effects are mediated through activation of peroxisome proliferator-activated receptor .alpha. (PPAR.alpha.). However, DHEA can affect the expression of genes independently of PPAR.alpha., including the gene for the major inducible drug and xenobiotic metabolizing enzyme, cytochrome P 450 3A23. To elucidate the biochem. assocd. with DHEA treatment, we employed a cDNA gene expression array using liver RNA from rats treated with DHEA or the classic peroxisome proliferator nafenopin. Principal components anal. identified 30 to 35 genes whose expression was affected by DHEA and/or nafenopin. Some were genes previously identified as PPAR-responsive genes. Changes in expression of several affected genes were verified by quant. reverse transcriptase-polymerase chain reaction. These included aquaporin 3, which was induced by DHEA and to a lesser extent nafenopin, nuclear tyrosine phosphatase, which was induced by both agents, and 11.beta.-hydroxysteroid dehydrogenase 1, which was decreased by treatment with DHEA in a dose-dependent fashion. Regulation of 11.beta.-hydroxysteroid dehydrogenase 1 expression is important since the enzyme is believed to amplify local glucocorticoid signaling, and its repression may cause some of the metabolic effects assocd. with DHEA.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1

ACCESSION NUMBER: 2002:238656 BIOSIS
DOCUMENT NUMBER: PREV200200238656
TITLE: sgk: An essential convergence point for peptide and steroid hormone regulation of ENaC-mediated Na+ transport.
AUTHOR(S): Faletti, Carla J.; Perrotti, Nicola; Taylor, Simeon I.; Blazer-Yost, Bonnie L. (1)
CORPORATE SOURCE: (1) Dept. of Biology, Indiana Univ.-Purdue Univ. at Indianapolis, 723 W. Michigan St., SL 358, Indianapolis, IN, 46202: bblazer@iupui.edu USA
SOURCE: American Journal of Physiology, (March, 2002) Vol. 282, No. 3 Part 1, pp. C494-C500. http://www.ajpcon.org. print. ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English

AB To study the role of sgk (**serum**, **glucocorticoid** -induced **kinase**) in hormonal regulation of Na+ transport mediated by the epithelial Na+ channel (ENaC), **clonal** cell lines stably expressing **human** sgk, an S422A sgk mutant, or a D222A sgk mutant were created in the background of the A6 model renal epithelial cell line. Expression of normal sgk results in a 3.5-fold enhancement of basal transport and potentiation of the natriferic response to anti-diuretic hormone (ADH). Transfection of a S422A mutant form of sgk, which cannot be phosphorylated by phosphatidylinositol-dependent **kinase** (PDK)-2, results in a cell line that is indistinguishable from the parent line in basal and hormone-stimulated Na+ transport. The D222A sgk mutant, which lacks **kinase** activity, functions as a dominant-negative mutant inhibiting basal as well as peptide- and steroid hormone-stimulated Na+ transport. Thus sgk activity is necessary for ENaC-mediated Na+ transport. Phosphorylation and activation by PDK-2 are necessary for sgk stimulation of ENaC. Expression of normal sgk over endogenous levels results in a potentiated natriferic response to ADH, suggesting that the enzyme is a rate-limiting step for the hormone response. In contrast, sgk does not appear to be the rate-limiting step for the cellular response to aldosterone or insulin.

L5 ANSWER 4 OF 11 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002068840 EMBASE

TITLE: Gene expression and identification of gene therapy targets in diabetic nephropathy.
AUTHOR: Wada J.; Makino H.; Kanwar Y.S.
CORPORATE SOURCE: Dr. J. Wada, Department of Medicine III, Okayama University Medical School, 2-5-1 Shikata-cho, Okayama 700-8558, Japan. junwada@meews1.med.okayama-u.ac.jp
SOURCE: Kidney International, (2002) 61/SUPPL. 1 (S73-S78).
Refs: 35
ISSN: 0085-2538 CODEN: KDYIA5
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A number of novel genes that are upregulated in diabetic kidneys have been identified. Recently, transforming growth factor-.beta. (TGF-.beta.)-driven secreted proreins, i.e., connective tissue growth factor (CTGF) and gremlin, were identified. They are up-regulated in kidneys of diabetic animals and modulate the biology of mesangial cells. CTGF mediates TGF-.beta.-induced matrix overproduction by the mesangial cells. Gremlin is a putative antagonist of bone morphogenetic protein-2 that blocks mesangial cell proliferation. Thus, gremlin may modulate the biology of mesangium by stimulating mesangial cell proliferation and in turn production of matrix. In addition, transcriptionally regulated **kinases**, **serum glucocorticoid**-regulated **kinase** and munc-13 have been identified. The former stimulates renal tubular Na(+) transport and is involved in hyperfiltraion of diabetic kidneys by a Na(+) transport feedback mechanism. Munc-13 has been shown to induce apoptosis in hyperglycemic state via diacylglyecrol-activated, PKC-independent signaling pathway. Another pathway relevant to diabetic nephropathy is polyol pathway, where glucose is reduced to sorbitol by aldose reductase. Recently, a renal-specific reductase of the aldo-keto reductase family was **isolated**. It is up-regulated in diabetic mice, and this could serve as a suitable target for gene therapy in renal complications of diabetes. Several mitochondrial genome-encoded genes, such as, cytochrome oxidase and NADH dehydrogenase, are up-regulated in diabetic kidneys. A novel nuclear-encoded mitochondrial gene, i.e., translocase inner mitochondrial membrane 44 (Tim44), is up-regulated in diabetic kidneys, and it may also serve as another target for molecular therapeutic intervention at the core storage energy sites, i.e., mitochondria. In this review, these novel differentially regulated genes that respond to hyperglycemic stress are described, and they may serve as possible targets for gene therapy in the treatment of diabetic nephropathy.

L5 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:85705 CAPLUS
DOCUMENT NUMBER: 138:366014
TITLE: K+ channel activation by all three isoforms of serum- and glucocorticoid-dependent protein **kinase** SGK
AUTHOR(S): Gamper, N.; Fillon, S.; Feng, Y.; Friedrich, B.; Lang, P. A.; Henke, G.; Huber, S. M.; Kobayashi, T.; Cohen, P.; Lang, F.
CORPORATE SOURCE: Department of Physiology, University of Tuebingen, Tuebingen, 72076, Germany
SOURCE: Pfluegers Archiv (2002), 445(1), 60-66
CODEN: PFLABK; ISSN: 0031-6768
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The serum- and glucocorticoid-dependent **kinase** SGK1 was

originally identified as a glucocorticoid-sensitive gene. Subsequently, the two homologous **kinases** SGK2 and SGK3 have been **cloned**, being products of distinct genes, which are differentially expressed and share 80% identity in amino acid sequence in their catalytic domains. While SGK1 has been shown to activate ion channels, including K+ channels, the functions of SGK2 and SGK3 have not been examd. The present study was therefore performed to elucidate the effect of SGK1, SGK2, and SGK3 on elec. properties of renal epithelial cells. To this end **human** embryonic kidney (HEK293) cells were transfected with the **kinases** and ion-channel activity detd. using the patch-clamp technique. In non-transfected cells and in cells transfected with the empty GFP construct a voltage-gated K+ current was obsd. amounting to 303.+-.19 pA (n=13) and 299.+-.29 pA (n=23), resp. Transfection with SGK1, SGK2 or SGK3 increased the voltage-gated K+ current to 1056.+-.152 pA (n=17), 555.+-.47 pA (n=17), and 775.+-.98 pA (n=16), resp. The K+ current was fully blocked by 3 mM tetraethylammonium chloride and inhibited 45% by the Kv1 channel blocker margatoxin (10 nM). In dual electrode voltage-clamp expts. SGK isoforms up-regulated Kv1 voltage-gated K+ channels expressed in Xenopus laevis oocytes. The present observations thus reveal a powerful stimulating effect of all three isoforms of SGK on K+ channels. Those effects may participate in regulation of epithelial transport, cell proliferation, and neuromuscular excitability.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:421165 CAPLUS
DOCUMENT NUMBER: 133:68896
TITLE: Activating serum and glucocorticoid-induced protein **kinase** and drug screening
INVENTOR(S): Cohen, Philip; Kobayashi, Takayasu; Deak, Maria
PATENT ASSIGNEE(S): The University of Dundee, UK
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000035946	A1	20000622	WO 1999-GB4232	19991214
W: JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1141003	A1	20011010	EP 1999-961205	19991214
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002533063	T2	20021008	JP 2000-588203	19991214

PRIORITY APPLN. INFO.: US 1998-112217P P 19981214
GB 1999-19676 A 19990819
WO 1999-GB4232 W 19991214

AB A method of activating serum and glucocorticoid-induced protein **kinase** (SGK) is provided wherein the SGK is phosphorylated. The SGK may be phosphorylated by PDK1 and/or a prepn. contg. PDK2 activity. A method of identifying a compd. that modulates the activity of SGK is provided, wherein the activity of SGK is measured by measuring the phosphorylation by SGK of a polypeptide comprising an amino acid sequence corresponding to the consensus sequence (Arg/Lys; preferably Arg)-X-(X/Arg)-X-X-(Ser/Thr)-Z wherein X indicates any amino acid, X/Arg indicates any amino acid, with a preference for arginine, and Z indicates that the amino acid residue is preferably a hydrophobic residue. The SGK may be activated by phosphorylation. The invention relates to screening methods for finding new drugs or lead compds.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:42532 CAPLUS
DOCUMENT NUMBER: 130:106933
TITLE: A **human** homolog of the rat **serum** **glucocorticoid**-regulated **kinase** and a cDNA encoding it
INVENTOR(S): Kumar, Sanjay; Zou, Cheng
PATENT ASSIGNEE(S): Smithkline Beecham Corporation, USA
SOURCE: Eur. Pat. Appl., 27 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 889127	A1	19990107	EP 1998-304830	19980618
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CA 2235785	AA	19990101	CA 1998-2235785	19980623
JP 11123086	A2	19990511	JP 1998-186223	19980701
US 2001027184	A1	20011004	US 2001-784249	20010215

PRIORITY APPLN. INFO.: US 1997-51446P P 19970701
US 1997-997212 A 19971223

AB H-SGK2: a **human** homolog of the rat **serum** **glucocorticoid**-regulated serine/threonine **kinase** is identified and a cDNA encoding it is **cloned**. The protein may be of use in the treatment of a no. of diseases (no data). Preliminary identification of the cDNA was made by searching EST databases for members of the serine/threonine protein **kinase** family. A pair of partial overlapping **clones** were identified and primers derived from them were used to obtain a full-length cDNA. The gene was found to be expressed in the hippocampus, osteoblasts, and dendritic cells.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 11 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000038163 MEDLINE
DOCUMENT NUMBER: 20038163 PubMed ID: 10569806
TITLE: Differential gene expression in tumorigenic and nontumorigenic HeLa x normal **human** fibroblast hybrid cells.
AUTHOR: Tsujimoto H; Nishizuka S; Redpath J L; Stanbridge E J
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics, University of California-Irvine College of Medicine, Irvine, California, USA.
CONTRACT NUMBER: CA 19401 (NCI)
CA 39316 (NCI)
SOURCE: MOLECULAR CARCINOGENESIS, (1999 Dec) 26 (4) 298-304.
Journal code: 8811105. ISSN: 0899-1987.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF160757
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991228

AB Fusion of tumorigenic HeLa cells with **human** skin fibroblasts

results in chromosomally stable hybrids that are nontumorigenic and no longer express the HeLa tumor-associated marker intestinal alkaline phosphatase (IAP). Previous studies of spontaneous tumorigenic segregants from the nontumorigenic hybrids implicated the loss of one copy of **human** fibroblast chromosome 11 in the concomitant reexpression of tumorigenicity. In an attempt to identify genes involved in the control of tumorigenic expression, we performed differential display screening of nontumorigenic hybrid cells and tumorigenic segregants. Subsequent northern blot analyses reproducibly showed 17 differentially expressed genes, eight of which were expressed differentially in the nontumorigenic hybrids and nine of which were expressed differentially in the tumorigenic hybrids. The former were genes for 80K-L protein (a substrate of protein **kinase** C), AXL/UFO (a receptor tyrosine **kinase**), insulin-like growth factor binding protein 3, apolipoprotein AI regulatory protein, collagen type I alpha-2 chain, transforming growth factor-beta-induced gene product 3 (BIGH3), pregnancy-specific beta-1-glycoprotein, and fibroblast activation protein alpha. The latter nine genes were genes for **serum/glucocorticoid** -regulated **kinase** (SGK; a serine/threonine protein **kinase**), PTPCAAX1 (a tyrosine phosphatase), CXCR-4 (a G-protein-coupled membrane receptor), L-kynurenine hydrolase, beta-1, 4-galactosyltransferase, keratin 8, keratin 17, and H19 and a novel gene. The differential expression of these genes provided several interesting candidates for regulation of tumorigenic expression, including those involved in signal transduction and the extracellular matrix, cytoskeletal proteins, cell-surface enzyme, and the H19 gene.

L5 ANSWER 9 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3

ACCESSION NUMBER: 2000:49948 BIOSIS
DOCUMENT NUMBER: PREV200000049948
TITLE: **Cloning** and mapping of a novel **human** **serum/glucocorticoid** regulated **kinase**-like gene, SGKL, to chromosome 8q12.3-q13.1.
AUTHOR(S): Dai, Fangyan; Yu, Long (1); He, Hua; Zhao, Yong; Yang, Jun; Zhang, Xianning; Zhao, Shouyuan
CORPORATE SOURCE: (1) Institute of Genetics, Fudan University, 220 Handan Road, Shanghai, 200433 China
SOURCE: Genomics, (Nov. 15, 1999) Vol. 62, No. 1, pp. 95-97. ISSN: 0888-7543.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB **Serum/glucocorticoid** regulated **kinase** (sgk) belongs to a newly emerging subfamily of the serine/threonine protein **kinase** family. Although **human** SGK shares 98% amino acid identity with rat sgk, their expression levels are regulated differently, which indicates the existence of other SGKs in **humans**. In this paper, we reported the **cloning** of **human** SGKL, which encodes a protein sharing 67 and 66% amino acid identity with rat sgk and **human** SGK, respectively. A 4.4-kb transcript of **human** SGKL was detected in 16 **human** tissues examined and was found to be most abundant in lung. By radiation hybrid mapping, the SGKL gene was located to **human** chromosome 8q12.1-q13.1 between markers D8S510 and D8S1797.

L5 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:604791 CAPLUS
DOCUMENT NUMBER: 129:213510
TITLE: The **human** homolog of the cell volume regulated protein **kinase** sgk and the gene encoding it
INVENTOR(S): Lang, Florian; Waldegger, Siegfried

PATENT ASSIGNEE(S): Dade Behring Marburg G.m.b.H., Germany
SOURCE: Eur. Pat. Appl., 15 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 861896	A2	19980902	EP 1998-101338	19980127
EP 861896	A3	19991020		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
DE 19708173	A1	19980903	DE 1997-19708173	19970228
CA 2224404	AA	19980828	CA 1998-2224404	19980226
US 6326181	B1	20011204	US 1998-31295	19980226
JP 10248566	A2	19980922	JP 1998-46565	19980227
US 2003003559	A1	20030102	US 2001-39	20011204

PRIORITY APPLN. INFO.: DE 1997-19708173 A 19970228
US 1998-31295 A3 19980226

AB The **human** gene for the cell vol.-regulated **kinase** sgk (serum and glucocorticoid-dependent **kinase**) is **cloned** and characterized. The enzyme can be used in the diagnosis and treatment of diseases assocd. with abnormal changes in cell vols. or macromol. crowding. Genes induced in HepG2 cells under hypertonic and hypotonic conditions were identified by RAP-PCR. A specific transcript that was expressed under hypertonic and hypotonic conditions was further characterized. The sequence of the full-length transcript had a 95% identity to a part of the rat sgk gene.

L5 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:647919 CAPLUS
DOCUMENT NUMBER: 125:294014
TITLE: Identification by subtractive hybridization of a spectrum of novel and unexpected genes associated with in vitro differentiation of **human** cytotrophoblast cells
AUTHOR(S): Morrish, D. W.; Linetsky, E.; Bhardwaj, D.; Li, H.; Dakour, J.; Marsh, R. G.; Paterson, M. C.; Godbout, R.
CORPORATE SOURCE: Department Medicine, University Alberta, Edmonton, AB, T6G 2S2, Can.
SOURCE: Placenta (1996), 17(7), 431-441
CODEN: PLACDF; ISSN: 0143-4004
PUBLISHER: Saunders
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We have previously demonstrated that epidermal growth factor (EGF), colony stimulating factor-1 (CSF-1), and granulocyte-monocyte colony stimulating factor (GMCSF) stimulate, while transforming growth factor .beta.1 (TGF.beta.1) inhibits, cytotrophoblast differentiation. To identify genes mediating EGF-induced differentiation, we constructed a subtracted cDNA library between undifferentiated cytotrophoblast and differentiating cytotrophoblast. We identified six novel genes and four known syncytial products .alpha.-**human** chorionic gonadotrophin (.alpha.hCG) pregnancy-specific .beta.1-glycoprotein, 3.beta.-hydroxysteroid dehydrogenase, and plasminogen activator inhibitor type 1 whose mRNAs increased during differentiation. Ten other genes were identified whose mRNAs increased during differentiation. Five of these (keratin 19, calreticulin, heat shock protein 27, serum and glucocorticoid-regulated **kinase** and adrenomedullin) were not previously reported to be expressed in placenta. Five other genes known to be expressed in placenta were identified: keratin 8, fibronectin, mitochondrial ATP synthase, H19, and cytosolic copper-zinc superoxide dismutase (SOD-1). Several of these

genes may have regulatory functions in trophoblast differentiation.

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WEST

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L4	L3 same (purif$n or isolat$n or character$n or clon$n)	3	L4
L3	L2 same human	17	L3
L2	L1 same (serum glucocorticoid)	28	L2
L1	kinase	43645	L1

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☐ 1. Document ID: US 20030003559 A1

L4: Entry 1 of 3 File: PGPB Jan 2, 2003

PGPUB-DOCUMENT-NUMBER: 20030003559
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030003559 A1

TITLE: Cell volume-regulated human kinase h-sgk

PUBLICATION-DATE: January 2, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Lang, Florian	Tubingen		DE	
Waldegger, Siegfried	Tubingen		DE	

US-CL-CURRENT: 435/194; 435/320.1, 435/325, 435/69.1, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6416759 B1

L4: Entry 2 of 3 File: USPT Jul 9, 2002

US-PAT-NO: 6416759
DOCUMENT-IDENTIFIER: US 6416759 B1
**** See image for Certificate of Correction ****

TITLE: Antiproliferative Sgk reagents and methods

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 6326181 B1

L4: Entry 3 of 3 File: USPT Dec 4, 2001

US-PAT-NO: 6326181
DOCUMENT-IDENTIFIER: US 6326181 B1

TITLE: Cell volume-regulated human kinase h-sgk

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